"STUDY DRUGS": THE MECHANISMS OF ADHD MEDICATIONS AND THEIR ABUSE ON COLLEGE CAMPUSES

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I. INTRODUCTION

The term "study drugs" refers to a sub-class of prescription stimulants, approved by the FDA for the treatment of Attention Deficit/Hyperactivity Disorder (ADHD), that students routinely abuse to enhance academic performance. This sub-class, categorized as substituted phenethylamines, includes methylphenidate (Ritalin, Concerta), dexmethylphenidate (Focalin), dextroamphetamine (Dexedrine), lisdexamfetamine (Vyvanse), and a mixture of amphetamine salts (Adderall). In the past decade, illicit abuse (i.e., without a prescription) of study drugs on college campuses has skyrocketed, with studies estimating that up to thirty-five percent of college students abuse such stimulant medications.[1]

Remarkably, study-drug abuse, unlike the abuse of alcohol and other recreational drugs, fails to elicit condemnation among parents and medical professionals,[2] who frequently dismiss this behavior as benign and of meager concern. For example, when asked about stimulant abuse

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[1] Jason Besser, *Do We Have an Amphetamine Problem on College Campuses*, CTR. ON ADDICTION (Oct. 3, 2017), https://www.centeronaddiction.org/the-buzz-blog/do-we-have-amphetamine-problem-college-campuses [https://perma.cc/ZR5F-BY28].

[2] Amelia M. Arria & Robert L. DuPont**,** *Nonmedical Prescription Stimulant Use among College Students: Why We Need To Do Something and What We Need To Do*, J. ADDICT DIS. 29(4) 417, 418 (2010), https://www.tandfonline.com/doi/full/10.1080/10550887.2010.509273 [https://perma.cc/QKP4-HRHM].

among college students, Dr. Brian Doyle, a clinical professor of psychiatry at Georgetown University Medical Center, gave the following response:

> It's like the psychological equivalent of using steroids to enhance physical performance. These students seem to be doing it with relative impunity, and it doesn't seem to be causing too much trouble since most use the drugs not to get high but to function better. So when exams are over, they go back to normal and stop abusing the drugs.[3]

Unconcerned views like Dr. Doyle's underscore the extent to which study drugs have become a normalized part of college life. This explainer will first discuss the underlying biochemistry and physiological effects of study drugs. Thereafter, the evolution of study drug abuse will be examined.

II. HOW STUDY DRUGS AFFECT THE BODY AND BRAIN

A. Neurotransmitters: The Brain's Biochemical Messengers

Neurotransmitters are chemical messengers, which transmit signals between neurons (specialized cells in the brain and nervous system) and from neurons to muscle cells.[4] Neurons are connected to other neurons by synapses, which include the extremities on either side of the neuron, and the microscopic gap between the cells through which chemical signals pass, known as the synaptic cleft.[5] Generally, an electric stimulus, called an action potential, travels down the length of a neuron on one side of the synapse (the pre-synaptic neuron), triggering the release of neurotransmitters from the synaptic vesicles (neurotransmitter storage sacs in the neuron) into the synapse.[6] On the other end of the synapse, the discharged neurotransmitters will interact with specific receptors on the receiving (post-synaptic) neuron.[7] Each neurotransmitter has a designated

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[3] Mathew Shulman, *Ritalin and the Risk of Substance Abuse*, U.S. NEWS (Mar. 3, 2008, 5:45 PM), https://health.usnews.com/health-news/family-health/articles/2008/03/03/ritalin-and-the-risk-of-substance-abuse [https://perma.cc/UUF8-FLY8].

[4] *What Are Neurotransmitters?*, QUEENSLAND BRAIN INST., https://qbi.uq.edu.au/brain/brain-physiology/what-are-neurotransmitters [https://perma.cc/3W5G-R34S].

[5] *Id.*

[6] *Action Potentials and Synapses*, QUEENSLAND BRAIN INST., https://qbi.uq.edu.au/brain-basics/brain/brain-physiology/action-potentials-and-synapses [https://perma.cc/9T9T-ZP45].

[7] *Id.*

receptor into which it fits, like a "lock and key," with only chemically compatible neurotransmitters attaching to a given receptor.

Once a neurotransmitter binds to a receptor, it causes the receiving neuron to respond by either firing an action potential (an excitatory response, which further propagates the signal) or by not firing an action potential (an inhibitory response, which stops the propagation of the signal).[8] After binding to receptors, neurotransmitters may (1) degrade or be deactivated by enzymes in the synapse, (2) drift away from the receptor into the synapse, and/or (3) undergo re-uptake, a process by which neurotransmitters return to the pre-synaptic neuron.

Study drugs interfere in the neurotransmission process, changing how effectively neurotransmitters are able to signal between cells. These interferences enable study drugs to influence the major physiological activities the nervous system regulates, including: heart rate, sleep, appetite, mood, breathing, and alertness. Study drugs predominantly exert their influence by interacting with a specific subclass of neurotransmitter—monoamines—which includes dopamine, norepinephrine, and serotonin.

Monoamine neurotransmitters are classified based on their molecular composition, exhibiting a single amino group (NH_2) linked by a two-carbon chain (CH_2-CH_2) to a benzene ring.[9] The structure of study drug molecules closely resembles monoamine neurotransmitters, meaning that they can bind to receptors, which would have otherwise been reserved for specific neurotransmitters.

Study drugs chiefly interfere in the neurotransmission of the monoamine, dopamine, and norepinephrine. Dopamine plays an essential role in motor control, information processing, motivation, reward, sexual gratification, reinforcement, alertness, attention, and endurance.[10] Norepinephrine is the brain's central mechanism for regulating the body's fight-or-flight response, alertness, memory formation and retrieval,

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[8] *Id.*

[9] Figure 1 displays the pertinent chemical groups of these compounds in red. *See* John R. Richards et al., *Methamphetamine, "Bath Salts," and Other Amphetamine-Related Derivatives: Progressive Treatment Update*, ENLIVEN ARCHIVE (Aug. 26, 2014), http://www.enlivenarchive.org/articles/methamphetamine-bath-salts-and-other-amphetaminerelated-derivatives-progressive-treatment-update.pdf [https://perma.cc/S2S4-FMD5] (source of Fig. 1).

[10] *What Are Neurotransmitters?, supra* note 4; *see* Kate Brophy, *What Is Dopamine? Understanding the "Feel-Good Hormone"*, UNH DAILY (Oct. 18, 2018), https://universityhealthnews.com/daily/depression/what-is-dopamine-understanding-the-feel-good-hormone/ [https://perma.cc/VZ8V-EC2C].

restlessness, and anxiety.[11] Figure 1[12] below displays the chemical structure of these two neurotransmitters, with their common chemical group outlined in red. Moreover, Figure 1 also presents a side-by-side comparison of dopamine and norepinephrine to several chemical agents which act upon their effectiveness, including the study drugs Adderall (amphetamine) and Ritalin (methylphenidate).

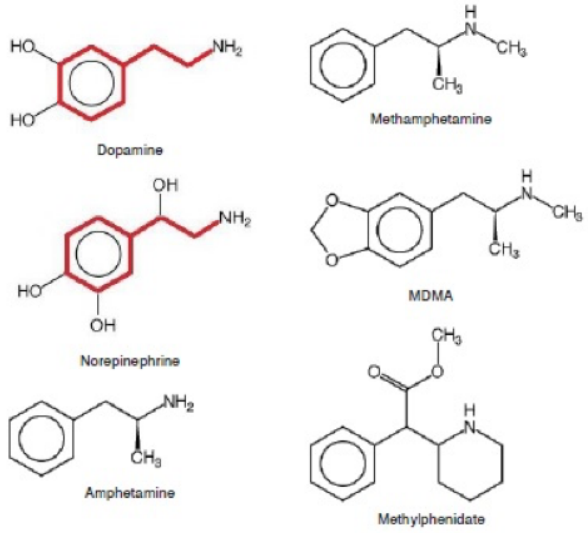

Figure 1. Chemical structure of dopamine, norepinephrine, methylphenidate, cocaine, amphetamine and related derivatives. The common β-phenylethylamine core structure is highlighted. Due to this structural similarity, amphetamines act as competitive substrates at plasmalemmal and vesicular membrane transporters of dopamine, norepinephrine, and serotonin, inhibiting reuptake and inducing reverse transport of these endogenous monoamines.

B. The Dopamine Deficit Hypothesis of ADHD

The precise causes of ADHD are unknown, but scientists theorize that the impaired functioning of certain neurotransmitter systems in the brain is at the crux of the disorder.[13] Evidence suggests that individuals with ADHD have an overly efficient dopamine-removal mechanism in the

[11] *See* Cathy Cassata, *What Is Norepinephrine?*, EVERYDAY HEALTH (Dec. 11, 2015), https://www.everydayhealth.com/norepinephrine/guide/ [https://perma.cc/GR7D-AKZZ]; *see also What Are Neurotransmitters?, supra* note 4.
[12] Richards et al., *supra* note 9.
[13] Margaret V. Austin, *Neurotransmitter Changes with ADHD*, GULF BEND CTR., https://www.gulfbend.org/poc/view_doc.php?type=doc&id=13861&cn=3 [https://perma.cc/N5AQ-8C5A].

area of the brain controlling motivation and reinforcement learning.[14] Excessive concentrations of dopamine reuptake inhibitors, the cellular transporters that return dopamine from the synapse back to the neuron which secreted it, remove dopamine from the synapse too quickly, before the neurotransmitter can bind to receptors and transmit its signal.[15] The result of the low dopamine signaling in the brain's reward and motivation centers is a diminished ability to concentrate and retain information—the major symptoms of ADHD.

Despite the soundness of this model as an explanation for ADHD, scientists have not yet found direct evidence of overactive dopamine reuptake in patients with ADHD. Ironically, the model is primarily premised on the effectiveness drugs, like Ritalin, have on relieving ADHD related symptoms.[16] During the 1950s and 1960s, clinicians began administering these stimulants to patients who reported attention and motivation deficits symptoms, and observed improvements in patients prescribed the medication. Subsequent experiments on model organisms determined that these stimulants produce increased dopamine activity in the brain's reward centers. Accordingly, the effectiveness of the treatment combined with the physiological effects demonstrated in model organisms led scientists to infer that dopamine deficiency was the underlying culprit behind ADHD pathology.

This uncertainty in ADHD's underlying mechanism creates a diagnostic quandary: what is the threshold of overactive dopamine reuptake at which an individual has ADHD categorically? ADHD cannot be revealed by blood test or brain scan. Instead, psychiatrists evaluate the manifestation of symptoms empirically, by watching patients, interviewing parents, and conducting questionnaires and psychoanalytical tests.[17] Furthermore, those who do not have ADHD report the same stimulant benefits—of increased concentration and motivation—as ADHD patients do. Indeed, a compelling body of research suggests that the stimulants prescribed to treat ADHD produce heightened attentiveness, working memory, and task saliency in normal adult subjects without ADHD.[18] Hence, the absence of quantitative testing methods for ADHD produces uncertainty arising from subjective testing.

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[14] *Id.*
[15] *Id.*
[16] *Id.*
[17] *Attention Deficit Hyperactivity Disorder: Diagnosing ADHD*, WEBMD (May 1, 2017), https://www.webmd.com/add-adhd/childhood-adhd/diagnosing-adhd#1.
[18] *See* Michael D. Devous et al., *Regional Cerebral Blood Flow Response to Oral Amphetamine Challenge in Healthy Volunteers*, 42 J. NUCLEAR MED. 535 (2001), http://jnm.snmjournals.org/content/42/4/535.long [https://perma.cc/8LY6-2NQ5].

C. Pharmacology of Common Study Drugs

Study drugs generally operate by increasing the activity of the neurotransmitters, primarily dopamine, in the brain.[19] Study drugs exploit their similarity in molecular structure to natural neurotransmitters (Figure 1) and compete with them for the binding sites of enzymes and receptors involved in their biochemical activity. Exploring the mechanism of action of three major classes of study drugs, amphetamines (Adderall), lisdexamfetamine (Vyvanse), and methylphenidate (Ritalin, Concerta) provides ample insight into how they produce the physiological sensations that make them so attractive to college students.

1. Amphetamine (Adderall)

Amphetamine's mechanism of action is the product of its structural similarity to the monoamine neurotransmitters, dopamine and norepinephrine.[20] These similarities allow amphetamine to compete to bind with monoamine reuptake transporters, NET (noradrenaline transporter) and DAT (dopamine transporter).[21] NET and DAT are protein structures embedded in the cell membrane of presynaptic neurons, responsible for the reuptake of their respective neurotransmitters from the synapse back into the neuron.[22] These transporters bind to their respective monoamine neurotransmitters and facilitate their return to the inside of the neuron.[23] Amphetamine molecules in the synapse compete with the endogenous monoamines for binding spots on the reuptake transporter. When an amphetamine molecule steals a spot on the reuptake transporter, it reduces the number of neurotransmitters transported back into the neuron.[24] Ultimately, the result is in an increased concentration of dopamine and norepinephrine in the synapse, allowing the neurotransmitters increased time and opportunity to bind to receptors and exert their activity on the receiving neuron and cause a transmission.

In addition to inhibiting reuptake, amphetamine also inhibits the action of synaptic monoamine oxidase (MAO), an enzyme (biological

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[19] *See* Austin, *supra* note 13.
[20] David J. Heal et al., *Amphetamine, Past and Present – a Pharmacological and Clinical Perspective*, 27(6) J. PSYCHOPHARMACOLOGY 479, 482 (2013), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3666194/pdf/10.1177_0269881113482532.pdf [https://perma.cc/ZN5Y-YE9A].
[21] *Id.*
[22] *Id.*
[23] *Id.*
[24] *Id.*

catalyst) that deactivates monoamine neurotransmitters in the synapse by "clipping off" their amine (NH_2) group.[25] Without an amine group, monoamine neurotransmitters are unable to bind to receptors and transmit signals to the receiving neuron. Similarly to how amphetamine competes with natural neurotransmitters for spots in reuptake transporters, amphetamine will also bind to MAOs in place of the natural neurotransmitter, reducing the quantity of free MAOs remaining in the synapse.[26] Therefore, more neurotransmitters remain active in the synapse and have a higher likelihood of transmitting a signal to the receiving neuron.

Amphetamine also exerts activity inside the pre-synaptic neuron itself.[27] The vesicular monoamine transporter 2 (VMAT2) transports newly synthesized monoamine neurotransmitters, located in the cytosolic pool (the fluid inside a cell), into synaptic storage vesicles within the neuron.[28] Amphetamine competes to bind to VMAT2 in place of the neurotransmitters. This initiates a process known as reverse transport, during which newly synthesized cytosolic monoamine neurotransmitters are pumped out of the cell and into the synapse, instead of, into the cell's intracellular storage vesicle.[29] The result of amphetamine's intracellular activity is a heightened potency in comparison to other study drugs.

2. Lisdexamfetamine (Vyvanse)

Vyvanse is another common ADHD medication and common study drug. Vyvanse is a pro-drug of dextroamphetamine, meaning it is a biologically inactive compound that is metabolized in the body to produce the actual drug.[30] Once the pro-drug is metabolized into its active

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[25] *Id*. *See generally Monoamine Oxidase*, SCI. DIRECT (2019), https://www.sciencedirect.com/topics/neuroscience/monoamine-oxidase [https://perma.cc/2KAM-V486] (providing a wealth of scientific articles explaining the mechanism and activity of the MAO enzyme).

[26] Heal et al., *supra* note 20.

[27] *Id.*

[28] *Id.*

[29] *Id.*

[30] *Lisdexamfetamine*, DRUGBANK (Feb. 9, 2019, 5:19 AM), https://www.drugbank.ca/drugs/DB01255 [https://perma.cc/AL6S-MZEY]; *see* William C. Shiel Jr., *Medical Definition of Prodrug*, MEDICINENET (Jan. 1, 2017), https://www.medicinenet.com/script/main/art.asp?articlekey=23992 [https://perma.cc/UHX8-CMP7] (defining pro-drugs).

amphetamine form, its mechanism of action is identical to other amphetamines such as Adderall.[31]

Vyvanse, though it produces the same effects as amphetamines, has a more complex structure than amphetamine drugs and is more expensive. The intended benefit accompanying its cost is that it is less susceptible to abuse than the pure amphetamine drug[32] (though it may still be abused as a study drug). Unlike pure amphetamines, Vyvanse exerts its effects gradually throughout the day, requiring only a single dose.[33] This is due to the pro-drug also producing the amino acid L-lysine as it metabolizes, which delays the progression of amphetamine through the blood stream and nervous system.[34] Additionally, Vyvanse will produce minuscule effects if ingested inappropriately via methods such as ingestion through the nostrils because the pro-drug requires gastrointestinal enzymes to metabolize into amphetamine.[35] Because snorting the drug will bypass this metabolic process, snorting will not transform the drug into amphetamine, making it less likely to be abused as a stimulant.

3. Methylphenidate (Ritalin and Concerta)

Methylphenidate was the first stimulant approved by the FDA in 1955.[36] Like amphetamines, it increases dopamine and norepinephrine activity by competing with the neurotransmitters for the binding sites on monoamine reuptake transporters. However, Methylphenidate is purely a reuptake inhibitor; unlike amphetamines, it does not inhibit the deactivation of neurotransmitters through MAO or block the storage of neurotransmitters through VMAT2.[37] The consequence of methylphenidate's narrower biochemical functionality is a reduced potency compared to amphetamines. More precisely, ten milligrams of the

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[31] *See Lisdexamfetamine*, *supra* note 30.
[32] *Id*
[33] *Id.*
[34] *Id.*
[35] *See id.*
[36] *See All Approvals December 1955*, FDA (Apr. 18, 2019), https://www.accessdata.fda.gov/scripts/cder/daf/index.cfm?event=reportsSearch.process&rptName=1&reportSelectMonth=12&reportSelectYear=1955&nav [https://perma.cc/7LST-76UN].
[37] *See* L.E. Arnold, *Methylphenidate vs. Amphetamine: Comparative Review*, 3(4) J. ATTENTION DISORDERS 200, 200 (2000), https://pdfs.semanticscholar.org/c4e7/f487339c67a2947472630f75a07e593c2950.pdf [https://perma.cc/8JPJ-JPL2].

brand name methylphenidate, Ritalin, is functionally equivalent to five milligrams of Adderall, the market-leading brand name amphetamine.[38]

D. Physiological Effects of Study-Drugs

Irrespective of the difference in mechanisms of action between sub-types, the net result of study drug consumption is increased dopamine and norepinephrine activity in the brain. Due to the colossal range of physiological activities the nervous system regulates through these neurotransmitters, stimulants can generate complex and myriad effects on users.

The immediate effects of study drugs are desirable to students and ADHD patients alike: increased motivation attached to relevant activities, heightened concentration, an amplified pleasure response, and mild euphoria.[39] While perhaps an undesirable side effect for ADHD patients, the energy and insomnia associated with these stimulants appeals to students as they prepare to work long hours or stay awake through the night.[40]

Study drugs also have additional adverse cardiovascular, neurological, and gastrointestinal effects associated with elevated activity in the nervous system. Cardiovascular effects include hypertension (high blood pressure), increased heart rate, and irregular heartbeat.[41] Neurological side-effects may include anxiety, headaches, decreased sexual arousal, dizziness, aggressive behavior, appetite loss, hallucination, and mild tics.[42] Gastrointestinal effects include stomach aches, constipation, diarrhea, and weight loss.[43] Additionally, users commonly report dry mouth and teeth-grinding.[44] Nonetheless, stimulants are relatively safe at recommended medical dosages, with most users reporting only mild undesirable effects from the drugs.[45]

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[38] For a detailed comparison of the relative potencies of various ADHD medications, see generally Matt Swenson, *Stimulant Equivalency Table*, UACAP, http://www.uacap.org/uploads/3/2/5/0/3250432/stimulant_equivalency.pdf [https://perma.cc/Z923-EW38].

[39] *The Effects of Amphetamine Use*, DRUGABUSE.COM (Dec. 3, 2018), https://drugabuse.com/amphetamine/effects-use/ [https://perma.cc/SN72-BX42].

[40] *See id.*

[41] *Id.*

[42] *Id.*

[43] *Side Effects of Adderall*, AM. ADDICTION CTRS. (Feb. 7, 2019), https://americanaddictioncenters.org/adderall/side-effects [https://perma.cc/S23Z-BYSR].

[44] *Id; The Effects of Amphetamine Use*, *supra* note 39.

[45] *See* Henry A. Spiller et al., *Overdose of Drugs for Attention-Deficit Hyperactivity Disorder: Clinical Presentation, Mechanisms of Toxicity, and Management*, 27 CNS

At high dosages, study drugs carry a risk of overdose.[46] An overdose is typically associated with extremely high or abnormal heart rate, hypertension, confusion, difficulty breathing, and muscular tremors.[47] A collective manifestation of these side-effects may be deadly if not treated promptly.[48] Nevertheless, national stimulant overdose fatalities are rare, especially in comparison to other drugs.[49]

Study drug abuse may lead to addiction. As users develop a tolerance for these drugs, they may increase their dosage to the point where the likelihood of devolving addiction increases substantially.[50] Also, as study-drug abusers become accustomed to the cognitive boosts that study-drugs deliver, they may become increasingly unable or unwilling to engage in many activities without the aid of study drugs.[51] As research explains, relying on study-drugs may discourage students from developing sustainable study habits, analogously to how reliance on dieting pills often leads consumers to avoid developing long-term weight-loss habits like regular exercise and healthy eating.[52]

III. THE ABUSE OF STUDY DRUGS ON COLLEGE CAMPUSES

Study drugs have become ubiquitous with college life, with as many as thirty-five percent of college students claiming to have used study drugs for non-medical objectives.[53] The widespread and normalized use of study drugs among college students raises questions as to how students gain access to these drugs and why they engage in their use.

A. Diagnostic Uncertainty and the Diversion of Legitimately Prescribed ADHD Medications Ensures a Steady Supply of Study Drugs

ADHD is the most commonly diagnosed mental health disorder in the U.S., with over ten percent of the entire U.S. population between the

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DRUGS 531 (2013), https://doi.org/10.1007/s40263-013-0084-8 [https://perma.cc/2JFB-UJE2]; *see also Side Effects of Adderall*, *supra* note 43.

[46] *See* Spiller et al., *supra* note 45.

[47] *See id.*

[48] *See id.*

[49] *See id.*

[50] *The Effects of Amphetamine Use, supra* note 39.

[51] *See* Arria & DuPont, *supra* note 2 (describing how dependence can lead to the development of poor self-discipline).

[52] *Id.*

[53] This statistic excludes the millions of ADHD diagnosed students who use stimulant medications legally. Besser, *supra* note 1.

ages of seven and twenty-five currently afflicted.[54] This diagnosis rate represents a five-fold increase in national ADHD cases since 1990. From 1993 to 2001, Adderall production alone has increased 5,767 percent, and world-wide revenue from all ADHD stimulants has exploded from $1.7 billion in 2002 to $9 billion in 2012.[55] Up to twenty-nine percent of students with legitimate stimulant prescriptions report that their peers have solicited them to give, sell, or trade their medication, and as many as sixty-two percent of college students with prescriptions divert at least part of their supply to non-prescribed students.[56] Thus, rising ADHD medication prescription rates ensure that study drugs are readily available on campus.

The dramatic rise of ADHD patients in the U.S. can be attributed to the subjective clinical diagnostic methodology used when testing for the disorder.[57] There is no objective "normal" amount of dopamine activity from which a negative deviation can be conclusively characterized as ADHD. Instead, ADHD is diagnosed in accordance with the Diagnostic and Statistical Manual, Fifth Edition guidelines (DSM-V).[58] The guidelines consist of two sets of criteria: (1) inattention and (2) hyperactivity and impulsivity. Each of these categories provides a list of symptoms which are indicative, but not alone definitive, of ADHD.[59] When a mental health professional concludes that the requisite number of these symptoms are found, she is authorized to categorize the patient as having ADHD and to prescribe stimulants as treatment.

The test for ADHD relies on vague criteria, such as an inability to focus on tasks, fidgeting, or interrupting others that are also common

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[54] *See generally Data & Statistics*, CDC (Sept. 21, 2018), https://www.cdc.gov/ncbddd/adhd/data.html [https://perma.cc/H8J6-T6NV]. This page contains all the most recent ADHD related statistics as gathered by the Center for Disease Control and Prevention.

[55] Besser, *supra* note 1.

[56] Arria & DuPont, s*upra* note 2; Shaheen E. Lakhan & Annette Kirchgessner, *Prescription Stimulants in Individuals with and without Attention Deficit Hyperactivity Disorder: Misuse, Cognitive Impact, and Adverse Effects*, 2 BRAIN & BEHAVIOR 661, 665 (2012), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3489818/pdf/brb30002-0661.pdf [https://perma.cc/ASF7-9HQ4].

[57] *See generally* C. Thomas Gualtieri & Lynda G. Johnson, *ADHD: Is Objective Diagnosis Possible?*, 2 PSYCHIATRY 44, 44 (2005), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2993524/pdf/PE_2_11_44.pdf [https://perma.cc/7ENM-TJJZ].

[58] For an in-depth look at the DSM-V guidelines for ADHD diagnosis, see *Symptoms and Diagnosis*, CDC (Dec. 20, 2018), https://www.cdc.gov/ncbddd/adhd/diagnosis.html [https://perma.cc/K7QL-GAWC].

[59] This list includes enumerations such as "often has trouble holding attention on tasks or play activities," "is often easily distracted," and "often avoids, dislikes, or is reluctant to do tasks that require mental." *Id.*

among adults and children who are not affected by ADHD.[60] Moreover, clinicians, in conjunction with parents who value academic performance,[61] have adopted a liberal approach to ambiguous ADHD diagnostics, erring on the side of stimulant-based treatment.[62] Indeed, studies have found that the average time it takes a patient to obtain a positive ADHD diagnosis can be as little as fifteen minutes and that up to twenty percent of ADHD patients may be misdiagnosed.[63]

The murky nature of ADHD diagnostics enables people without ADHD to receive medication by simulating the symptoms.[64] Dedicated adults can quite easily deceive a medical professional into believing they have ADHD. As many as ninety-three percent of people succeed in fooling their psychiatrists into prescribing them amphetamine medications.[65] Cassie Schwartz, a renowned author in the field of developmental neuroscience and psychoanalysis, shared her account of how she obtained an Adderall prescription while a student at Brown:

> I went to the nearest campus computer and searched for 'cognitive behavioral psychiatrist' . . . . The very next day, I was . . . describing to the young psychiatrist how I had always had to develop elaborate compensatory strategies for getting through my school work, how staying with any one thing was a challenge for me, how I was best at jobs that required elaborate multitasking, like waitressing. Untrue, all of it. I was a focused student and a terrible waitress. And yet these were the answers that I discovered from the briefest online research were characteristic of the A.D.H.D. diagnostic criteria. These were the answers they were looking for in order to pick up their pens and write down 'Adderall, 20 mg, once a day' on their prescription pads. So these were the answers I gave. Fifty minutes later, I was standing . . . prescription slip in hand.[66]

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[60] Sanford Newmark, *Are ADHD Medications Overprescribed?*, WALL ST. J. (Feb. 14, 2013, 9:20 PM), https://www.wsj.com/articles/SB10000872396390444301704577631591596516110.
[61] Arria & DuPont, *supra* note 2.
[62] *See* Newmark, *supra* note 60.
[63] *Id.*
[64] Kelly Lord, *How Adderall Became Overprescribed and Underestimated*, ELITE DAILY (Dec. 13, 2016), https://www.elitedaily.com/wellness/adderall-overprescribed-underestimated/1709645 [https://perma.cc/56CU-K3X7].
[65] *Id.*
[66] Casey Schwartz, *Generation Adderall*, N.Y. TIMES (Oct. 12, 2006), https://www.nytimes.com/2016/10/16/magazine/generation-adderall-addiction.html [https://perma.cc/UUS9-UQDH].

Schwarz easily obtained an Adderall prescription by deliberately feigning the pertinent ADHD symptoms specified in the DSM-V. The conclusions of researchers corroborate Schwarz's account, finding that for an adult who is determined to gain legal access to ADHD medications, success is very likely.[67]

Not only can healthy patients feign ADHD, but they can also receive prescriptions from multiple doctors simultaneously.[68] While HIPAA laws require physicians to maintain centralized patient records documenting the medications they prescribe, many neglect to reexamine these records when prescribing new medications.[69] Bad actors can legally procure a steady supply of study drugs as long as they can find doctors who will prescribe them medication.

Some psychiatrists voluntarily adopt more vigorous diagnostic safeguards to reduce the frequency of ADHD misdiagnosis. These safeguards include extending the length of the sessions and requiring patients to meet with them multiple times before rendering a diagnostic decision.[70] Additionally, the psychiatrist will closely monitor the patient after prescribing a stimulant prescription and periodically reassess whether the patient is benefiting from the prescribed medication and using it as intended.[71] However, these amplified procedures are not universally followed, and patients are free to "shop" for clinicians with a reputation for quick and liberal stimulant prescription practices.[72]

ADHD is a chronic mental health condition that can be unduly burdensome if left untreated.[73] To many suffering from ADHD, treatment with stimulants is critical to the enjoyment of their daily lives and normal functioning.[74] Nevertheless, current ADHD diagnostic methods are vague and easily exploited, ensuring that a legal supply of study drugs is unlikely to subside.

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[67] *See* Lord, *supra* note 64.
[68] *Id*.
[69] *See id.*
[70] *Id*.
[71] *Id*.
[72] A simple Google search of "how to get an Adderall prescription" reveals forums filled with people recommending doctors with a reputation for stimulant prescriptions.
[73] *See* Lord, *supra* note 64.
[74] *Id*.

B. Study Drugs Are in Demand Among Students Because of their Perceived Benefit During Studying

Study-drugs are perceived by many students as educational doping, permitting students to prolong study sessions by enhancing concentration, attention, motivation, and wakefulness.[75] Moreover, study drugs induce euphoria and help students relieve stress factors inherent to the educational environment.[76] Abuse rates of study drugs tend to peak during exam periods when students stay up to "cram" for tests and meet impending deadlines on assignments.[77] In general, the majority of students report that abusing study drugs has been helpful to their academic progress.[78] Notwithstanding, a growing body of research suggests that study drugs do not provide a pure boost to a healthy student's cognitive performance.[79]

Even though study drugs improve studying stamina by keeping students awake, concentrated, and euphoric, these heightened feelings do not necessarily translate into an increased intellectual capacity.[80] A study that examined student performance on reading comprehension and short-term memory tests, found no benefit from study drug ingestion.[81] Another study found that working memory enhancement from study drugs was correlated with an individual's baseline academic performance, with students who tend to perform well on intellectual tasks benefiting the least from study drugs.[82] Furthermore, studies have found that study drugs do not increase IQ.[83] Thus, study drugs, despite their boost to attentiveness and motivation, are not likely impacting baseline cognitive performance.

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[75] *See* Lakhan & Kirchgessner, *supra* note 56.

[76] *See generally* Steve Sussman et al., *Misuse of "Study Drugs:" Prevalence, Consequences, and Implications for Policy*, SUBSTANCE ABUSE TREATMENT, PREVENTION, & POL'Y (June 2006), https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1524735/pdf/1747-597X-1-15.pdf [https://perma.cc/4BNT-RYH6] (giving a detailed account of the effects of stimulants which promote their abuse among students).

[77] *Id*; Lakhan & Kirchgessner, *supra* note 56.

[78] Lakhan & Kirchgessner, *supra* note 56.

[79] *See id.*

[80] *See id*.

[81] David Rettew, *Does Adderall Make You Smarter?*, PSYCHOL. TODAY (July 27, 2018), https://www.psychologytoday.com/us/blog/abcs-child-psychiatry/201807/does-adderall-make-you-smarter [https://perma.cc/A7GN-QXNC].

[82] Lakhan & Kirchgessner, *supra* note 56.

[83] *Id*.

The demand for study drug consumption by students is powered by their desire to increase productivity, motivation, and concentration.[84] The universal efficacy of study drugs means that they are advantageous to both students who are struggling to get good grades and to those who are determined to outshine their fellow classmates. Despite data suggesting that study drugs do not increase pure intellectual performance, i.e. they do not make students smarter, the euphoria, sleeplessness, and increased attention they stimulate will likely continue to fuel the demand among students.

C. Study Drug Abuse Avoids Public Scrutiny and Is Perceived as Low-Risk by Students

The prevalence of study drug abuse can be further attributed to the neutral stance taken by society towards illicit study drug consumption and to the low risk many associate with study drug-abuse.[85] A study on media coverage of study drug abuse revealed that ninety-five percent of articles mentioned at least one benefit of study drug abuse, but only fifty-eight percent disclosed the risks or side-effects.[86] Academics say that parents use academic success to justify the abuse of these stimulants:

> [Parents appear] to be enabling the problem by turning a blind eye or even encouraging the behavior. Fueled by their concerns about maximizing their child's academic performance, these parents are highly susceptible to [believing] that, at best, nonmedical use of prescription stimulants might help their child earn better grades, and that, at worst, it is harmless.[87]

Many clinicians share a similar sentiment, seeing study drug abuse as a minor concern, since it is used for a positive aim—studying—and because abuse will likely recede after exams are complete.[88] Many students

[84] "A web-based survey administered to medical and health profession students found that the most common reason for nonprescription stimulant use was to focus and concentrate during studying." *See* Lakhan & Kirchgessner, *supra* note 56.
[85] *Id.*
[86] *Id*.
[87] Arria & DuPont, *supra* note 2.
[88] *Id.*

perceive study drugs as posing only a mild health risk, especially those who have previously experimented with the drugs.[89]

Study drugs have managed to circumvent active public disapproval and have failed to incite fear of any serious health risks from users, parents, or clinicians. In fact, the data suggests that the prevailing sentiment is one of passive acceptance.

IV. CONCLUSION

Study drug abuse is a prodigious phenomenon on college campuses across the nation. The ease of accessibility of study drugs coupled with the euphoric and motivational boost they provide users leads scores of students to reach for the "magic" pills in their pursuit of academic excellence.

[89] Indeed, students who have experimented with study-drugs consider the risk negligible. *See* Besser, *supra* note 1.